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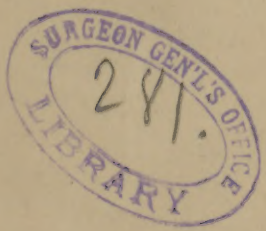

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A SUMMARY OF HOLSTI'S VIEWS ON ARTERIO-CAPILLARY SCLEROSIS.*

By F. B. STEPHENSON, M. D.,
UNITED STATES NAVY.

The original of this article † contains about thirty-five pages, and authorities for opinions quoted are given in numerous foot-notes.‡ The author opens his paper by citing and reviewing the statements of various writers on this disease, which is known also under the names *granular atrophy of the kidney*, *arterio-capillary fibrosis*,* *Bright's disease*, etc. He sums up this *critique* by saying that there are two principal opinions among observers, the chief point in discussion being the connection between lesions of the kidney and disease of the vascular system, especially of capillaries and smaller arteries. Some view the renal disease as primary,

* Read before the U. S. Naval Medical Society.

† Translated and summarized from "Om Förändringarna af de finare Arterna vid den granulära Njuratrofin och deras betydelse för denna sjukdoms Patologi." Af Hugo Holsti, Docent vid Universitetet i Helsingfors. "Nordiskt mediciniskt Archiv," sjuttonde Bandet. Första Häftet. 1885.

‡ Dr. Holsti is careful to state that he founds his judgments in this investigation not upon reports, but upon his own observed cases.

* Historic data on this subject may be found in an article by Dr. A. L. Loomis, under the title, "Arterio-capillary Fibrosis," printed in the "New York Medical Journal," February 20, 1886.

and the vascular change as a result thereof; while others think that certain forms of granular atrophy of the kidney may be brought about by such sclerosis, which, however, is not limited to the renal vessels, but may spread over the whole arterial system.

Hugo Holsti, the author, intimates that this disease has been considered too much in a histologic way, whereas its study from a clinical aspect might be more fruitful.

The data for his paper were furnished by twenty cases, nearly all observed in the medical clinic of the University of Helsingfors.

The earlier stages of the disease cause so slight a disturbance of the general health that they usually pass unnoticed. An increased amount of urine first attracts the patient's attention. On examination, the chief thing that the physician can find is a more or less disturbed action of the heart, with hypertrophy; the pulse is ordinarily hard, resistant; the arterial walls may be thickened and abnormally bent or curved.

Although these cardio-vascular symptoms seem to be the whole sickness, yet, as Traube advises, whenever we find hypertrophy of the heart without valvular lesion, we should suspect renal atrophy, and examine the urine.

This disease may be latent ten years or more, according to the physiological state and hygienic surroundings of the person. Sooner or later, gradually or suddenly, often with some affection of slight importance in itself, a change for the worse takes place. The patient becomes lean, sallow, or yellow; retinal trouble may be found; symptoms before named become more marked. Even in this condition, however, appropriate means for building up of the general health—good food, fit exercise, etc.—are able to bring about great improvement.

Toward the last, symptoms attributed to the retention

of certain constituents of the urine are observed. Constant headache, disordered sensation, œdema, dyspnœa, diarrhœa, apathy, hæmorrhage,* spasms, gradual stupor, coma, or an intercurrent acute disease in the lungs, pleura, etc., may conduct the sick one slowly to his end; an epileptoid seizure may quickly close the scene.

From the foregoing we may see that there are two well-marked divisions in the progress of this disease—one, cardio-vascular; the other, uræmic.

After this clinical consideration, the author gives the results of his examination into the pathologic anatomy as to heart-overgrowth, and the histologic change in the larger and smaller arteries. The abdominal aorta was sometimes sclerosed; but the greatest change appeared in the vessels of less and least size. The different components of the vascular walls were affected in an unequal degree. Often the intima was most involved; in other instances the muscular layer or the adventitia suffered, the remaining parts being comparatively healthy. Microscopic examination showed the walls of some small vessels so altered that they were a mere homogeneous mass, without distinction of structure.

This morbid condition was spread over the entire arterial system, although its intensity varied in different parts. It seemed quite marked at the bifurcation of the abdominal aorta, and in the coronary arteries; the pia mater was frequently affected.

Occasionally one kidney appeared relatively little changed, when in the other atrophy had reached the advanced stage known as "the small red kidney"; this the author describes in detail.

We find a table of measurements showing the diverse amounts of thickening undergone by the vascular walls.

* This may occur in the skin and mucous membrane, simulating the so-called hæmorrhagic diseases, or diatheses.

"Since we have seen, on the one hand, the clinical forms under which granular renal atrophy advances, and, on the other, the anatomic change coming therewith, it remains for us to try to make clear the very complicated question concerning the result and the mutual dependence of this complex of disease arising from widespread vascular sclerosis, cardiac hypertrophy, and wasting of the kidney."

Agreeing with Gull and Sutton, Dr. Holsti believes that the vascular change is the primary affection, giving rise to the others, and he does so "chiefly because the clinical progress of the disease is thus best explained." "In hypertrophy from different cause, as valvular lesion, we do not find polyuria." The headache, dizziness, rush of blood to the head, and hæmorrhages in the brain are easily comprehended as consequences of the sclerosis and hypertrophy.

The overgrown heart is long able to withstand the resistance of the hardened vessels with diminished lumen; but a time comes when the cardiac action is insufficient, over-fullness of blood in the lungs takes place, and asthma. Although this symptom may become prominent in arterio-capillary sclerosis, valvular heart trouble from other source is possible without it. Sometimes asthma occurs so early in the disease that it does not seem due to uræmia; it may also be caused by hardening and thickening of the coronary arteries.

Dyspeptic affections are better understood by the change in various branches of the abdominal aorta, as the parts they supply thus lose more or less of their customary nourishment; hence may arise anæmia, with possibly resulting ulceration, necrosis, etc. We have here, doubtless, the origin of gastric ulcer, and of so-called albuminuric retinitis.

An example of the peculiar logic used by some writers is found on the twenty-second page of the original Swedish article.

After referring critically to the theories and expressions of Thiry, Traube, Cohnheim, and others, our author concludes that we can not explain the heart's hypertrophy by disease of the kidney, either by mechanical obstruction in the vascular system thereof, or by action due to hæmic changes of renal cause; an inflammatory sclerosis of the small arteries gives rise to the cardiac enlargement and to the kidney disease.

Reference is made to the diagnosis of atheroma from renal cirrhosis. The former nearly always occurs in old people, is located especially in the larger arteries, and shows fatty and chalky matters on histological examination, whereas the latter is met with in persons about thirty years of age (two of his cases being under twenty), affects the smaller arteries more and earlier than the larger, and is an inflammatory process. Syphilis and lues are compared in like manner. This differentiation closes with the statement that the peculiar sclerosis found in granular atrophy of the kidney is not seen in any other malady.

More than seventy per centum of cases properly belonging to this disease have been incorrectly reported as affections of the brain, heart, and lungs. Renal disorder of other origin may exist quite independently.

In the opinion of our author, this cardio-vascular sclerosis, with renal atrophy, is caused by some irritant circulating in the blood and acting on the walls of the vessels, secondary lesions being produced in the kidneys, chiefly during excretion.* As a rule, the intima is first affected, but the outer layers do not escape. The adventitia, by reason of its histologic structure, is very apt to suffer. This

* "We know, by experiment, that we can bring about like changes first in the blood, and secondarily in the kidneys, through the introduction into the organism of such substances as cantharidin, turpentine, petroleum, etc."—Hugo Holsti.

inflammatory change is greatest where the blood rests longest in contact with the parts, as in the small arteries and capillaries, more particularly throughout the branches of the abdominal aorta and their vascular connexes. The changes both in the vessels and in the heart are due to previously morbid blood. The lessened capacity of distal vessels, from their thickened walls, brings on increased pressure in the larger arteries, and compensatory cardiac hypertrophy. The sclerosis in the general circulation and in the kidneys may progress at the same time without any necessary interdependence, each arising from like cause. So long as this hypertrophy and an abnormal permeability of renal vessels allow excretion of the natural constituents of the urine, the general health may be little if any disturbed. When the heart or the kidney fails, the uræmic stage begins.

From a comparative study of the symptoms and of the necroscopic condition, it results that prognosis is ordinarily somewhat better in acute uræmia than in chronic.*

This disease may be hereditary, or come about through the influence of personal surroundings more or less under man's control.

The facts brought to our knowledge in this paper have an evident bearing on certain habits of diet and modes of life.

* "Af det föregående framgår äfven, att den akuta uremin i allmänhet lemnar en något bättre prognos, ty den kan inträda, innan ännu njuratrofin skridit altför långt; den kroniska uremin antyder däremot alltid, att förstörelsen af njurväfnaden skridit till den yttorsta grad, och när denna därföre en gång fullständigt etablerat sig, är icke någon förbättring mera att vänta."—Hugo Holsti.

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